Case report: Alcoholic fatty liver associated with low alcohol intake

https://doi.org/10.56238/levv15n39-086

Aline Lourdes Pasqualli[1], Raphaela lorrana Rodrigues Araújo[2], Priscila Gabriella Carraro Merlos[3], Fernando Merlos[4].

ABSTRACT

Alcoholic liver disease is common in patients with increased alcohol intake, as a result of which liver damage and injury occurs that can impair proper functioning and metabolization in the liver. The objective of this study is to present the case of a female patient who developed alcoholic fatty liver disease, but with low alcohol intake. The patient presented laboratory and imaging tests with significant alterations, and some hypotheses were raised for differential diagnoses, following the investigation, a biopsy was performed with a favorable result for alcoholic hepatic steatosis. Thus, it is of great importance to recognize that females reach higher alcohol concentrations when compared to males, in addition to having a hormonal issue involved.

Keywords: Hepatic Steatosis, Liver, Female.

[1] Estácio College - IDOMED of Jaraguá do Sul
[2] Hospital Municipal São José
[3] Hospital Regional Hans Dieter Schmidt
[4] Hospital Regional Hans Dieter Schmidt

INTRODUCTION

Alcoholic liver disease is a consequence of excessive alcohol consumption that is directly linked to damage to the body, the liver participates in alcoholic oxidation, characterized by first-pass metabolism is the organ most susceptible to injury[1]. It performs several functions, including regulation of cholesterol and coagulation factors, production of proteins and enzymes, metabolic functions and detoxification[2].

Men can ingest a greater amount of alcohol when compared to women, it is known that around 20% of men who consume amounts greater than 80 g of ethanol and women about 20 g of ethanol daily, for a period of more than 10 years have a greater chance of developing liver cirrhosis[2].

Chronic alcohol use is considered in men who consume amounts greater than 40 grams/day, while for women more than 20 g daily is needed to be considered individuals with chronic use of alcoholic beverages. Alcoholic liver disease occurs in about 20% of patients considered alcoholics, who abuse it[3].

The present article aims to report the case of a young patient diagnosed with alcoholic hepatic steatosis associated with low alcohol intake, which in most patients requires increased consumption of alcoholic beverages to develop the disease. In view of this, it is important to know that even in young patients with a history of low alcohol intake, cases of steatosis may occur, advising and warning that even young patients should moderate the use of alcohol and take care of their health, as cases of steatosis may evolve in the future to hepatitis, cirrhosis and even cancer.

CASE REPORT

A 35-year-old female was referred to the emergency room after presenting hemoglobin of 3.5 in an external laboratory test. The patient had already been under investigation for anemic and weight loss for more than 1 year. He reported a history of recent hospitalization requiring 4 CHADS and the presence of long-term hemorrhoidal disease. At the time of admission, she reported asthenia, adynamia and jaundice that had started about two weeks earlier, in addition to laboratory tests with altered hemoglobin. On physical examination, the patient presented jaundiced, tense abdomen, no pain on palpation, but the presence of a palpable liver, suggesting hepatomegaly, no signs of ascites and peritonitis, and perimalleolar edema in the lower limbs showed progression of improvement. The patient was then submitted to laboratory and imaging tests that showed negative serology, bilirubin of 10, enlarged PAT of 1.8, LDH 325, TGP of 34, AST 155, GGT 1074 and FA 281 abdominal tomography corroborated hepatomegaly and hepatic fat deposition, upper digestive endoscopy with presence of moderate pangastritis and colonoscopy with findings of cecum colitis, without major particularities in the exams presented. In addition, following the investigation, peritoneal fluid was collected, which was negative for neoplastic cells, doppler of hepatic vessels and transvaginal USG,

all within normal standards. In view of this, the possibility of a possible autoimmune hepatitis/cholangitis was raised, and markers and liver biopsy were requested. Finally, liver biopsy showed a large extent of alcoholic fatty liver disease. However, the patient reported low alcohol intake, denied psychiatric events such as anorexia or bulimia, which make the case of hepatic steatosis with low alcohol intake.

DISCUSSION

The human body is able to metabolize the alcohol consumed in order to avoid accumulations and serious health dysfunctions, so there are several steps with the objective of degrading this substance through enzymes. After ingestion, it undergoes first-pass metabolism, usually in the gastric mucosa by alcohol dehydrogenase. A large amount of alcohol is absorbed through the intestinal route, traveling through the bloodstream until it reaches the liver through the portal system, being metabolized mainly in the hepatic route4.

Genetic factors are involved in the pathogenesis of alcoholic liver disease. The variety of enzymes responsible for chemical reactions that act on ethanol and acetaldehyde (AA) are directly linked to the emergence of dependence. Some genes implicated, especially those of alcohol dehydrogenase, play a lower metabolization of ethanol, favoring the pathway that generates metabolites that are aggressive to the liver. In addition, different forms of aldehyde dehydrogenase are related to greater sensitivity to alcohol in women and Asian peoples. Orientals who have dysfunction of ALDH2, an enzyme, are more likely to have liver damage. Thus, the patient in the aforementioned case presents the female gender as a possible coadjuvant for a greater predisposition to harmful effects of alcohol, which configures that women are more prone to liver damage[2].

Women may develop the disease in lower amounts than men and have worse prognosis. By ingesting the same amount of alcohol that males consume, they reach higher concentrations in the bloodstream, mainly because they have less body mass, less body water, causing less alcohol distribution. Estrogen actively participates by increasing intestinal permeability to endotoxins with greater expression of CD14 receptors, stimulating pro-inflammatory cytokines[2].

Females are especially vulnerable when compared to males with regard to liver damage involved in alcohol consumption. This can be explained by the fact that female first-pass metabolism is decreasing and has lower activity of cytoplasmic alcohol dehydrogenase of hepatocytes, an enzyme involved in metabolization. In addition, estrogen increases the risk of female liver damage and higher levels of ethanol in the systemic and portal circulation are also expected [3,5]. The patient in the study fits into this context because she is a woman and has higher levels of circulating estrogen, mainly because she is in a young, reproductive age group, as mentioned, there are greater chances of

hepatic harmful effects, which corroborates the above, as the young woman opened the clinical picture with alcoholic hepatic steatosis.

Patients present with characteristic laboratory abnormalities that draw attention to possible diseases. As for hepatic steatosis, there is a significant increase in aminotransferases, especially an AST/SGT ratio greater than 3, indicating a factor suggestive of alcoholic liver disease, especially in those who do not have cirrhosis. When performing laboratory analysis of the patient, she presented a level higher than 3, corroborating the AST/AST ratio and a possible diagnosis of alcoholic hepatic steatosis[2].

CONCLUSION

The article brings a relevant theme, mainly due to the harmful effects of alcohol and because it is legally inserted in society and more and more young people come into contact with alcoholic beverages. The present study demonstrates that even small amounts and low alcohol intake can be harmful, which calls attention to greater health care. Therefore, the research becomes a means of alerting young people who believe that alcohol will not cause a problem, because the intake is low, so the article brings clinical and scientific data that even low doses can lead to liver problems, alerting to be careful with the consumption of alcoholic beverages.

REFERENCES

Dani, R., & Passos, M. do C. F. (2018). Gastroenterologia essencial (4th ed. [Reprint]). Guanabara Koogan.

Moya, L. C. (2022). Spectrum of alcoholic liver disease: A review of pathophysiology and clinical repercussions. Brazilian Journal of Health Review, 5(4), 13904–13927. https://doi.org/10.34119/bjhrv5n4-159. Available at: https://ojs.brazilianjournals.com.br/ojs/index.php/BJHR/article/view/50944/38439

Dukić, M., Radonjić, T., Jovanović, I., Zdravković, M., Todorović, Z., Kraišnik, N., Arandelović, B., Mandić, O., Popadić, V., Nikolić, N., et al. (2023). Alcohol, inflammation, and microbiota in alcoholic liver disease. International Journal of Molecular Sciences, 24(4), 3735. https://doi.org/10.3390/ijms24043735. Available at: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC9966185/

Teschke, R. (2019). Alcoholic liver disease: Current mechanistic aspects with focus on their clinical relevance. Biomedicines, 7(3), 68. https://doi.org/10.3390/biomedicines7030068. PMID: 31491888; PMCID: PMC6783919. Available at: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6783919/

Liu, S. Y., Tsai, I. T., & Hsu, Y. C. (2021). Alcohol-related liver disease: Basic mechanisms and clinical perspectives. International Journal of Molecular Sciences, 22(10), 5170. https://doi.org/10.3390/ijms22105170. PMID: 34068269; PMCID: PMC8153142. Available at: https://pubmed.ncbi.nlm.nih.gov/34068269/